Shock

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Learning objective

✓ To understand what shock is, what causes it, and how it is best managed according to the cause.

Shock is characterized by inadequate perfusion of vital organs, principally the heart and brain.

Aetiology

Tissue perfusion requires an adequate blood pressure, which is dependent upon the systemic vascular resistance and cardiac output; the cardiac output is a function of the heart rate and the stroke volume. These may be expressed in mathematical
terms: $CO = HR \cdot SV$
$BP = CO \cdot SVR$
where CO is cardiac output, SV is stroke volume, HR is heart rate, BP is arterial blood pressure and SVR is systemic vascular resistance.

Normal regulation of tissue perfusion

The autonomic nervous system is able to alter heart rate and peripheral vascular resistance in response to changes in blood pressure detected by the carotid sinus and aortic arch baroreceptors; changes in systemic vascular resistance may alter venous return by changing the amount of fluid circulating in the cutaneous and splanchnic vascular beds.
Venous return determines stroke volume; increasing venous return causes an increase in stroke volume, the heart acting as a permissive pump (Starling ' s law: 1 the output depends on the degree of stretch of the heart muscle at the end of diastole). Volume regulation is achieved by the kidney, in particular by the regulation of sodium loss by the renin – angiotensin – aldosterone system and antidiuretic hormone (ADH) produced by the posterior pituitary; in addition, a fall in circulating volume prompts the sensation of thirst, stimulating increased fluid intake.

Abnormal regulation of tissue perfusion

Inadequate tissue perfusion (shock) may result from factors related to the pump (the heart) and factors relating to the systemic circulation. The causes of shock may be classified accordingly, as follows:
1 ***Cardiogenic shock.*** A primary failure of cardiac output in which the heart is unable to maintain adequate stroke volume in spite of satisfactory filling. Compensation involves an increase in heart rate and systemic vascular resistance, manifested clinically by a tachycardia, sweating (due to sympathetic nervous system outflow), pallor and coldness (due to cutaneous vasoconstriction). Causes include the following:
a massive myocardial infarction;
b pulmonary embolism;
c acute ventriculoseptal defect following myocardial infarction affecting the septum;
d mitral or aortic valve rupture;
e acute cardiac tamponade.

2 ***Fluid loss***. Reduction in circulating volume results in a reduction in stroke volume and cardiac output. Blood pressure is initially maintained as in cardiogenic shock, with increased sympathetic activity raising the peripheral vascular resistance leading to the clinical picture of a cold, clammy patient with a tachycardia. As volume losses increase, the blood pressure falls. In severe cases, the patient is confused or semiconscious. Causes include:
a haemorrhage, revealed or internal (e.g. ruptured aneurysm; bleeding into the bowel or around a closed fracture);
b burns, with massive loss of plasma and electrolytes;
c severe diarrhoea or vomiting, with fluid and electrolyte loss, particularly in colitis or pyloric stenosis;
d bowel obstruction, in which large amounts of fluid are sequestered into the gut, in addition to the losses due to vomiting;
e peritonitis, with large fluid losses into the abdomen as a consequence of infection or chemical irritation;
f gastrointestinal fistulae with fluid and electrolyte loss;
g urinary losses, e.g. the osmotic diuresis of diabetic ketoacidosis, or polyuria in resolving acute tubular necrosis.

3 ***Reduction in systemic vascular resistance***. Reduction in systemic vascular resistance increases the size of the systemic vascular bed, producing a relative hypovolaemia, reduced diastolic fi lling, reduced stroke volume and thus a fall in blood pressure. Unlike the previous two causes, vasodilatation occurs as part of the pathogenesis, so the patient appears warm (‘ hot shock ’), not cold and peripherally shut down. The heart compensates with an increase in output. The principal causes are:
a anaphylaxis;
b sepsis;
c spinal shock.

4 ***Confounding factors***. Pre - existing medical conditions and medications may confuse the clinical picture. Consider a patient, with hypertension and taking a β - blocker such as atenolol. For that patient, a systolic blood pressure of 110 mmHg may be very low, and the atenolol prevents a compensatory tachycardia in response.

Special causes of shock

Adrenocortical failure

Loss of the hormones produced by the cortex of the suprarenal gland may follow bilateral suprarenal haemorrhage, adrenalectomy, Addison ’ s disease 2 or lack of corticosteroid replacement in patients who have been on long – term glucocorticoids. Failure of aldosterone secretion results in volume depletion and glucocorticoid deficiency,
which impairs autonomic responses.
The ability to respond to minor stress is severely compromised and may provoke an Addisonian crisis characterized by bradycardia and postural hypotension, which is responsive to corticosteroid replacement. Adrenocortical failure should be considered and a bolus of hydrocortisone given in all patients with unexplained hypotension.

Sympathetic interruption

This reduces the effective blood volume by widespread vasodilatation. It follows transection of the spinal cord (spinal shock), but may also occur after a high spinal anaesthetic.

The vasovagal syndrome (faint)

The vasovagal syndrome is produced by severe pain or emotional disturbance. It is the result of reflex vasodilatation together with cardiac slowing owing to vagal activity. Hypotension is caused by a fall in cardiac output due to both bradycardia and reduced venous return; the latter the result of peripheral vasodilatation. Clinically, it is recognized by the presence of a bradycardia and responds to the simple measure of laying the patient flat with elevation of the legs.

Septic shock

Shock may be produced as the result of severe infection from either Gram - positive or, more commonly, Gram - negative organisms. The latter are seen particularly after colonic, biliary and urological surgery, and with infected severe burns. The principal effect of endotoxins is to cause vasodilatation of the peripheral circulation together with increased capillary permeability.
The effects are partly direct and partly due to activation of normal tissue inflammatory responses such as the complement system and release of cytokines such as tumour necrosis factor (TNF).
Disseminated intravascular coagulation (DIC) results from activation of the clotting cascade and may lead to blockage of the arterial microcirculation by microemboli.
Fibrin and platelets are consumed excessively, with resultant spontaneous haemorrhages into the skin, the gastrointestinal tract, the lungs, mouth and nose.

Sequelae of shock

A continued low blood pressure produces a series of irreversible changes, so that the patient may die in spite of treatment. The lack of oxygen affects all the vital organs.

- *Cerebral hypoperfusion* results in confusion or coma.
- *Renal hypoperfusion* results in reduced glomerular filtration, with oliguria or anuria.

As renal ischaemia progresses, tubular necrosis may occur, and profound ischaemia may lead to cortical necrosis.

- *The heart* may fail owing to inadequate coronary perfusion.
- *Pulmonary capillaries* may reflect the changes in the systemic circulation with transudation of fluid resulting in pulmonary oedema, hampering oxygen transfer and causing further arterial hypoxaemia and thus tissue hypoxia.

Pulmonary capillary function may also be impaired following multiple blood transfusions and contusions resulting from chest trauma, a condition known as acute lung injury (previously termed ‘ shock lung ’).

- *DIC* , precipitated by sepsis, may be further aggravated by hypothermia unless active re - warming is undertaken.

Comparison of different types of shock		
Hypovolemic	Hypotension, tachycardia Weak thready pulse Cool, pale, moist skin U/O decreased	Decreased CO **Increased SVR**
Cardiogenic	Hypotension, tachycardia Weak thready pulse Cool, pale, moist skin U/O < 30 ml/hr Crackles, tachypnea	Decreased CO **Increased** SVR
Neurogenic	Hypotension, BRADYCARDIA **WARM DRY SKIN**	Decreased CO **Venous & arterial vasodilation, loss sympathetic tone**
Anaphylactic	Hypotension, tachycardia Cough, dyspnea Pruritus, urticaria Restlessness, decreased LOC	Decreased CO **Decreased** SVR
Septic	Hypotension, Tachycardia Full bounding pulse, tachypnea **Pink, warm, flushed skin** Decreased U/O, fever	**Decreased** CO, **Decreased** SVR

Principles in the management of patients in shock

Immediate measures

The immediate treatment of patients in shock varies according to cause. Two causes merit mention for immediate treatment: bleeding and anaphylaxis.

Bleeding

Direct pressure should be applied to a bleeding wound. Immediate surgical exploration is indicated where continued bleeding is likely, such as in peptic ulcer haemorrhage, ruptured spleen, ruptured aortic aneurysm or ruptured ectopic pregnancy. In these cases, resuscitation cannot overcome the losses until the rate of blood loss is curtailed.

Anaphylaxis

In surgical practice, this may arise as an allergic reaction to an antibiotic or radiological contrast medium. In addition to hypotension (due to vasodilatation), bronchospasm and laryngeal oedema may be present and warrant immediate therapy. The immediate treatment for anaphylaxis is the administration of adrenaline (epinephrine; 0.5 mL of 1:1000 concentration)

intramuscularly or subcutaneously, repeated every 10 – 30 minutes as required. Subsequently, hydrocortisone and antihistamine agents may be given (e.g. chlorphenamine).
For milder reactions, aliquots of 1 mL of 1:10 000 adrenaline are given and titrated to effect.

Monitoring and subsequent management

The severely shocked patient should be admitted to an intensive care ward where continuous supervision by specially trained nursing staff is available. As well as careful clinical surveillance, the following need to be monitored:

- Core temperature, pulse, respiration rate and blood pressure.
- Hourly urine output (via a urinary catheter).
- Central venous pressure.
- Pulse oximetry. Oxygen is administered to ensure adequate oxygenation. Mechanical ventilation may be required.
- Electrocardiogram (ECG).
- Serum electrolytes, haemoglobin and white blood cell count.
- Arterial blood gases (*P* o2 , *P* co2 , [H +]).
- The cardiac output, and left atrial and pulmonary arterial pressures using a Swan – Ganz catheter.

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Prevention of hypothermia

Patients may cool down because of neglect, infusion of cold fluids, particularly unwarmed blood, and extracorporeal circulations such as haemodialysis or haemofiltration circuits. Allowing a patient to cool down to subnormal temperatures (35 ° C and below) impairs the coagulation cascades and platelet aggregation, and promotes fibrinolysis, possibly resulting in DIC. To prevent
this, all infusions should be prewarmed, and the patient actively re- warmed using warm air blankets.

Pharmacological a gents

The shocked patient may require significant pharmacological support. The principal drugs used are catecholamines or their derivatives, in addition to drugs to treat specific causes such as antimicrobial therapy for septicaemia. Patients in cardiogenic
shock benefit from positive inotropic agents, whereas patients with low systemic vascular resistance due to sepsis require agents to increase vascular resistance. The drugs used in this context are sympathomimetics, with differing degrees of α (peripheral vasoconstriction), β 1 (inotropic and chronotropic) and β 2 (peripheral vasodilatation) effects. Examples of such drugs include the following.

Dopamine

Dopamine has three separate actions according to
dose:

1-At *low doses* (2 μ g/kg/min) dopaminergic actions dominate, causing increased renal

perfusion. This is the commonest indication for the use of dopamine.
2- At *moderate doses* (5 μ g/kg/min), β1 effects predominate with positive inotropic activity (increasing myocardial contractility and rate).
3- At *higher doses* (over 5 μ g/kg/min), α effects predominate with vasoconstriction.

Dopexamine

Dopexamine has predominantly β 2 actions, increasing myocardial contractility; it also acts on peripheral dopamine receptors, increasing renal perfusion.

Dobutamine

Dobutamine has predominantly β 1 actions, increasing myocardial contractility and rate, thus increasing cardiac output. It is used principally in cardiogenic shock.

Noradrenaline (norepinephrine)

Noradrenaline has predominantly α effects, but with modest β activity. It is used to increase systemic vascular resistance through its vasoconstrictor α effects.

Adrenaline (e pinephrine)

Adrenaline has strong α and β actions, and may be used to increase peripheral resistance while also increasing cardiac output. The powerful vasoconstrictor actions of both adrenaline and noradrenaline may result in ischaemia and infarction of peripheral tissues, most commonly fi ngers, toes and the tips of the nose and ears.